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# AP Biology

## Sample Student Responses and Scoring Commentary

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# AP<sup>®</sup> BIOLOGY 2018 SCORING GUIDELINES

## Question 2

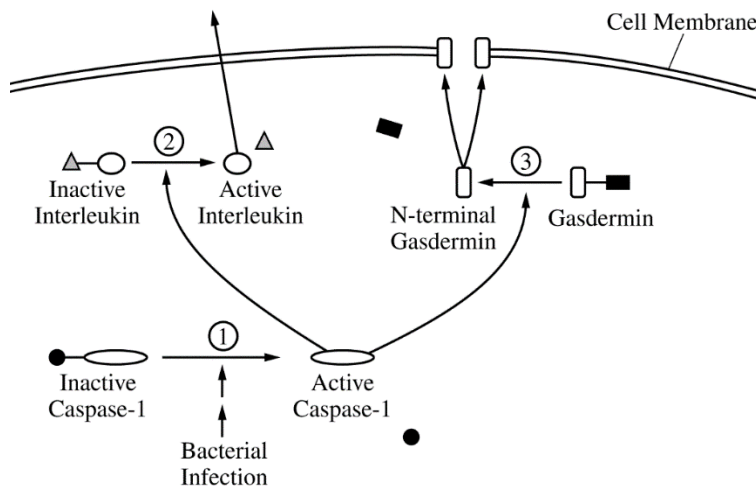


Figure 1. Cellular response to infection by pathogenic bacteria

Some pathogenic bacteria enter cells, replicate, and spread to other cells, causing illness in the host organism. Host cells respond to these infections in a number of ways, one of which involves activating particular enzymatic pathways (Figure 1). Cells normally produce a steady supply of inactive caspase-1 protein. In response to intracellular pathogens, the inactive caspase-1 is cleaved and forms an active caspase-1 (step 1). Active caspase-1 can cleave two other proteins. When caspase-1 cleaves an inactive interleukin (step 2), the active portion of the interleukin is released from the cell. An interleukin is a signaling molecule that can activate the immune response. When caspase-1 cleaves gasdermin (step 3), the N-terminal portions of several gasdermin proteins associate in the cell membrane to form large, nonspecific pores.

Researchers created the model in Figure 1 using data from cell fractionation studies. In the experiments, various parts of the cell were separated into fractions by mechanical and chemical methods. Specific proteins known to be located in different parts of the cell were used as markers to determine the location of other proteins. The table below shows the presence of known proteins in specific cellular fractions.

CELL FRACTIONS CONTAINING DIFFERENT CELLULAR PROTEINS

	Aconitase (Krebs cycle protein)	DNA polymerase	GAPDH (glycolytic protein)	Sodium- potassium pump	NF- $\kappa$ B (Immune response protein)
Whole cell sample	+	+	+	+	+
Fraction 1	+				
Fraction 2		+			+
Fraction 3			+		+
Fraction 4				+	
+ = presence of protein					

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**Question 2 (continued)**

(a) **Describe** the effect of inhibiting step 3 on the formation of pores AND on the release of interleukin from the cell.

**Description (2 points)**

- Pores will not form.
- Interleukin release will not be affected/interleukin release continues.

(b) **Make a claim** about how cleaving inactive caspase-1 results in activation of caspase-1. A student claims that preinfection production of inactive precursors shortens the response time of a cell to a bacterial infection. **Provide ONE reason** to support the student's claim.

**Claim (1 point)**

- Removes inhibitor/repressor/inhibitory domain of protein
- Changes the shape/protein structure

**Reasoning (1 point)**

- Cleaving a precursor/protein/molecule is faster than making one upon infection.
- Cells do not have to wait for transcription and translation/protein synthesis.

(c) A student claims that the NF- $\kappa$ B protein is located in the cytoplasm until the protein is needed for transcription. **Justify** the student's claim with evidence. **Identify TWO** fractions where N-terminal gasdermin would be found in cells infected with pathogenic bacteria.

**Justification (1 point)**

- NF- $\kappa$ B and glycolytic enzymes/GAPDH are found together (in the cytoplasm).

**Identification (2 points)**

- Fraction 3
- Fraction 4

(d) **Describe** the most likely effect of gasdermin pore formation on water balance in the cell in a hypotonic environment.

**Description (1 point)**

- Water enters the cell.

(e) **Explain** how gasdermin pore formation AND interleukin release contribute to an organism's defense against a bacterial pathogen.

**Explanation (2 points)**

- Cell lysis destroys infected cells OR cell lysis prevents bacteria from replicating.
- Interleukin signaling will stimulate immune cells/components of the immune system (to destroy the infected cells or bacteria).

2.a. Inhibiting step 3, the cleavage of caspase-1 by gadsolinin, by caspase-1, will prevent the association of gadsolinin proteins and the subsequent formation of pores in the cell membrane. This will not affect the release of interleukin from the cell because interleukin does not require the pores to leave the cell.

b. Cleaving inactive caspase-1 may alter the interaction of R-groups within the protein, resulting in a shape change in the cleaved molecule's tertiary structure that exposes an active site. If the cell did not constantly produce inactive caspase-1, the protein would have to be transcribed and translated before performing its function, a process which requires more energy and more time than a simple cleavage of a polypeptide.

c. <sup>and fraction 3, the cytosol</sup>  
 The student's claim is correct because fraction 2, the nucleus, is a tested positive for NF- $\kappa$ B protein. Fraction 2 is the nucleus because it contains DNA polymerase, an enzyme found only in the nucleus where DNA replication occurs and NF- $\kappa$ B would be needed to regulate transcription. Fraction 3 is the cytosol because this fragment tested positive for glycolytic proteins which aid in glycolysis, a process performed in the cytoplasm. N-terminal gadsolinin would be found in fraction 3 and fraction 4.

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## ADDITIONAL PAGE FOR ANSWERING QUESTION 2

d. In a hypotonic environment, water will move into the cell <sup>osmosis through</sup> via the pores because by definition, a hypotonic environment would have a higher water potential than the cell.

e. Hardermin pore formation causes the flow of water into the cell, which may cause the infected cell to burst. This would prevent the spread of the infection because phagocytes could not digest the components of the cell along with the pathogens. The release of interleukin activates the adaptive immune response by stimulating B and T leukocytes to divide and produce antibodies or kill infected cells, respectively.

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(a) By inhibiting step 3, caspase 1 won't end up cleaving gasdermin. This means the response of large nonspecific pores forming won't occur as there won't be N-terminal portions of gasdermin proteins to associate and form the pores in the cellular membrane.

By inhibiting the release of interleukin, the immune response can't be activated as interleukin is the signaling molecule. A consequence would be far spreading of the bacteria and illness.

(b) Cleaving inactive caspase-1 leads to active caspase 1 because it removes repressor proteins that maintain the inactive form of caspase-1.

One reason preinfection production of inactive precursors shortens response time is that ~~the~~ it reduces the number of steps/pathways to initiate cellular response. It won't be necessary to activate the genes in order to transcribe and translate the inactive precursors if they are already present.

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## ADDITIONAL PAGE FOR ANSWERING QUESTION 2

(c) Evidence that NF- $\kappa$ B remains in the cytoplasm until needed comes from fraction 2. ~~at~~ This is because GAPDH is a glycolytic protein that would be present in the cytoplasm to the cellular membrane. It wouldn't be in an organelle. As a result, ~~it would be present~~ <sup>the fraction 3</sup> indicates NF- $\kappa$ B isn't being used for transcription and would be located in the cytoplasm until needed. If ~~it wasn't~~ NF- $\kappa$ B isn't generated until transcription is taking place, then NF- $\kappa$ B wouldn't be in a fraction with GAPDH, a glycolytic protein. ~~It~~ NF- $\kappa$ B would only be observed near DNA such as in fraction 2.

N terminal gasdermin would be found in fractions 4 and 3 since these fractions are closest to the cellular membrane.

(d) In a hypotonic ~~environ~~ environment, gasdermin pore formation would allow water to move into the cell in a net ~~transfer~~ <sup>amount</sup> ~~transfer~~. This is because the cell would have a higher solute concentration than the surrounding environment and diffusion of water ~~is~~ <sup>involves moving</sup> towards higher solute concentration. ~~in order to decrease~~ This is regarded as osmosis.

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(e) Interleukin would contribute to a organism's defense by initiating an immune response. This may cause the release of white blood cells, helper T cells, and other species to limit bacterial infection

Gasdermin pore formation may help against the pathogen by allowing movement of many species in and out of the cell rather than through transport proteins. This may include signaling molecules that will initiate intracellular responses.

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a) By inhibiting step 3, the caspase-1 will not cleave gasdermin which will prevent the formation of the pores so interleukin will not be released from the cell.

b) Cleaving inactive caspase 1 could be removing an allosteric inhibitor that has binded to the enzyme causing shape change and stopping its function. Removing the inhibitor allows the enzyme to bind with another substrate such as the virus or other enzymatic proteins to create a cascade of events making it active.

One reason that supports the students claim is memory B cells which are an ~~an~~ immune response where the antibodies are already developed in the body and can be easily triggered to defend against bacteria. Since the immune response is already in the body and ready to be used, the trigger of bacteria entering the body will immediately generate the targeted response instead of waiting for the body to generate one from scratch.

c) NF- $\kappa$ B is found in the cytoplasm which is shown in Figure 2 which shows the fractions. Fraction 3 is the cytoplasm due to it containing other proteins and NF- $\kappa$ B starting out in the cytoplasm. This protein is also found in fraction 2 which contains DNA polymerase

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used for DNA replication in the nucleus. This shows that NF- $\kappa$ B is found in the cytoplasm and when needed moved to the nucleus because that is where transcription occurs since DNA polymerase is there for DNA replication. N-terminal gasdermin is in fraction 3 and 4 (cytoplasm and cell membrane).

d) The formation of the pore will cause an influx of water from the environment into the cell to restore water balance. Since the environment is hypotonic, there is not as much solute as in the cell, causing the water to diffuse into the cell through pore.

e) Interleukin is a signaling molecule that activates an immune response in the cell and will be released through the pores when formed. When it is released into the body it can bind to receptors on B and T cells which contain a specific immune response in order to generate antibodies to defend against the bacteria. The interleukin serves as the receptor protein that triggers the response of immune system cells to target, identify, and kill the bacteria.

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## 2018 SCORING COMMENTARY

### Question 2

#### Overview

This question required students to consider a cell signaling enzymatic pathway that involved caspase-1 in cells that had been infected by pathogenic bacteria. Background information was provided as a narrative and in the form of an illustration. Prior to infection, inactive caspase-1 precursors are formed that can be activated by cleaving the inactive caspase-1. Once activated, caspase-1 cleaves interleukin, an immune system cytokine, thereby activating it and allowing it to be released from the cell. Caspase-1 also cleaves gasdermin, which allows the N-terminal portion of the protein to form large pores in the plasma membrane. The students were provided with a diagram illustrating these steps. Students were asked to describe the impact of inhibiting gasdermin cleavage on pore formation and on interleukin release. Students made a claim about how inactive caspase-1 becomes activated by cleaving. They were further asked to provide reasoning for why having inactive caspase-1 precursors formed prior to infection shortens the response time after a cell is infected. In addition, the question contained a table that shows the location of five cellular proteins after fractionating the cell. Using these data, students justified a claim that the NF- $\kappa$ B protein is located in the cytoplasm of the cell. They identified two fractions where N-terminal gasdermin would be located based on both the image and information found in the table. Students were then asked to describe how the formation of gasdermin pores would affect water balance of cells in a hypotonic environment. Finally, students were asked to explain how gasdermin pore formation and interleukin release contribute to an organism's defense against a bacterial pathogen.

The key understandings and skills students were expected to demonstrate included the following:

- The scientific method and quantitative skills were used to interpret a diagram and data presented in a table.
- Knowledge of cell signaling, organelle function, molecular activation, osmosis and water potential, apoptosis, and specific components of the immune system were integrated and used to interpret a model.

#### Sample: 2A

#### Score: 10

The response earned 1 point in part (a) for describing that the effect of inhibiting step 3 will be to prevent the formation of pores. The response earned 1 point in part (a) for describing that interleukin release will not be affected. The response earned 1 point in part (b) for making the claim that cleaving will result in a shape change in the tertiary structure of the protein. The response earned 1 point in part (b) for providing reasoning that transcription and translation before cleavage take more time than simple cleavage of constantly produced inactive caspase-1. The response earned 1 point in part (c) for justifying the student's claim with evidence by stating that NF- $\kappa$ B is in the cytoplasm with glycolytic proteins used for glycolysis. The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 3. The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 4. The response earned 1 point in part (d) for describing that water moving into the cell was the most likely effect of gasdermin pore formation on water balance in the cell in a hypotonic environment. The response earned 1 point in part (e) for explaining that gasdermin pore formation contributes to an organism's defense against a bacterial pathogen because "... infected cells burst ... and prevent the spread of infection because phagocytes then digest the components of the cell along with the pathogens." The response earned 1 point in part (e) for explaining that interleukin release contributes to an organism's defense against a bacterial pathogen because it stimulates B and T leukocytes.

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**Question 2 (continued)**

**Sample: 2B**

**Score: 8**

The response earned 1 point in part (a) for describing that the effect of inhibiting step 3 is that pore formation will not occur. The response earned 1 point in part (b) for describing that cleaving inactive caspase-1 “removes repressor proteins.” The response earned 1 point in part (b) for explaining that the number of steps is reduced if the transcription and translation of inactive precursors do not have to occur because the precursors are already present. The response earned 1 point in part (c) by justifying the student’s claim with the evidence that NF- $\kappa$ B is found with GAPDH, a glycolytic protein, in the cytoplasm. The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 3. The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 4. The response earned 1 point in part (d) for describing that in a hypotonic environment, gasdermin pore formation “would allow water to move into the cell.” The response earned 1 point in part (e) for explaining that interleukin release would contribute to an organism’s defense against pathogens by causing the release of white blood cells and helper T cells.

**Sample: 2C**

**Score: 6**

The response earned 1 point in part (a) for describing that inhibiting step 3 will prevent the formation of pores. The response earned 1 point in part (b) for describing that cleaving inactive caspase-1 removes an allosteric inhibitor that was “causing shape change and stopping its function.” The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 3. The response earned 1 point in part (c) for identifying that N-terminal gasdermin would be found in Fraction 4. The response earned 1 point in part (d) for describing that pore formation would cause an influx of water. The response earned 1 point in part (e) for explaining that interleukin release would contribute to an organism’s defense against pathogens by binding “to receptors on B and T cells which contain a specific immune response.”